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Heat-stroke: a fatal case

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INTRODUCTION

Heat-stroke is a severe, life-threatening and increasingly common disease. Of all admissions to our ICU over the period from July 1981 to September 1984, 6·6% ($n = 605$) were for heat-related illness. The growing popularity of long-distance running in recent years has certainly contributed to an increased incidence of exertional heat-stress illness (Kains *et al.*, 1983).

CLINICAL DETAILS

Private J. P., a 17-year-old territorial army basic trainee, was brought to the casualty department at 07.25 hours on 18 July 1984. He was said to have collapsed and become unconscious 15 minutes into a 6 km march/run. At the time of the collapse he had been wearing light clothing and carrying around 12 kgs of equipment. There was nothing of note in his previous medical history. Collapse had taken place on the third morning of a 3-day exercise; opportunity for sleep would have been limited on the night before. A light meal had been provided at 03.00. At 06.00 the dry bulb temperature was 16·7°C and humidity 87%.

On arrival in casualty he was deeply unconscious with no response to pain. Rectal temperature was 40°C, pulse rate 140/minute, blood pressure 90/50, respiratory rate 60/minute and he was intensely peripherally constricted, the skin being cool and dry.

Intravenous infusion was commenced with 1 litre of 0·9% saline over the first half hour. As dextrostix on admission indicated a blood sugar of 1 mmol L^{-1}, 100 mls of 50% Dextrose were given immediately. He was stripped, tepid-sponged and given 30% oxygen by MC mask.

On transfer to ITU at 08.30 he remained deeply unconscious with marked limb rigidity and an oesophageal temperature of 42·8°C, pulse rate 155/min, BP 120/50, respiratory rate 60/min. He was paralysed (using vecuronium in view of the tachycardia), intubated and ventilated. Blood gas results and temperatures may be seen

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in Table 1. To reverse the severe metabolic acidosis, 200 mls 8·4% sodium bicarbonate were given. A urinary catheter was inserted and revealed a small amount of dark urine pH 5·0, with 3+ albumen, 1+ ketones and a trace of blood on stick testing. Blood chemistry may be seen in Table 2. ECG monitoring revealed sinus tachycardia with unifocal ventricular ectopic beats at a frequency of 4–6 per minute. He was producing copious watery diarrhoea.

Table 1 Blood gases

	Time					
	08.30	09.30	13.30	15.00	17.00	18.00
Temperature (rectal) °C	41·8	39·8	38·5	38·5	37·7	37·7
pH	7·246	7·403	7·439	7·442	7·434	7·364
pCO_2(kpa)	4·01	4·07	3·15	3·64	3·36	4·65
pO_2(kpa)	8·01	7·50	20·40	24·87	31·93	26·47
Saturation	73·5	88·9	99·1	99·4	99·7	99·4
HCO_3	11·8	18·8	15·6	18·1	16·7	19·4
TCO_2	12·5	19·7	16·3	18·9	17·5	20·4
Base excess	−13	−5	−7	−4·5	−6·5	−5
$^{F}I_{O_2}$	0·30	0·40	0·40 (ventilated)	0·35 (ventilated)	0·30 (ventilated)	0·30 (ventilated)

Cooling was continued by repeated tepid sponging and fanning and he was later covered by a Churchill thermo-regulating blanket through which cold water was circulating. He remained in intense peripheral vascular shut-down however, and cooling was slow despite a total of 50 mgs chlorpromazine given in 10 mg boluses in an attempt to produce enhanced cooling by peripheral vasodilation.

Radial arterial and internal jugular venous lines were inserted to facilitate direct pressure monitoring and repeated blood sampling. A Seldinger technique was thought particularly appropriate for the central line in view of the fact that, as early as 09.00 there was evidence of disseminated intravascular coagulation in the form of uncontrolled bleeding from puncture sites. A huge haematoma of the right groin was noted in relation to an earlier femoral arterial 'stab'.

Clotting screen revealed: Hb 14·3 g.dl^{-1}, APTT 85 seconds (n̄ 24–35), PTT 21·5 seconds (control 11·5), FDPS 160 < 320 ug.ml^{-1} (n̄ < 10). On haematological advice a total of 8 units of fresh frozen plasma were infused.

A total of only 130 ml urine were produced between admission and death despite a CVP maintained at + 10 cms H_2O and the infusion of 200 ml 20% Mannitol followed by dopamine 10 μgs/kg/min. Blood pressure fell to 70/50 around midday and never rose above this figure again. Cardiac arrest in asystole occurred at 19.00 hours and resuscitative efforts were unsuccessful.

Post-mortem revealed evidence of acute renal tubular damage and otherwise

Table 2 Blood chemistry (showing laboratory normal ranges)

	Time		
	09.00	11.30	13.25
Urea (3·1–6·1 mmol.L^{-1})	12·1	13·0	13·1
Creatinine (68–105 mmol.L^{-1})	238	256	264
Na^+ (138–145 mmol.L^{-1})	151	149	144
K^+ (3·3–4·5 mmol.L^{-1})	4·6	3·5	3·2
Cl (95–106 mmol.L^{-1})	115	115	110
TCO_2 (24–31 mmol.L^{-1})	17·7	18·9	21·8
Glucose (4·2–6·6 mmol.L^{-1})	4·7	9·6	10·5
Anion gap (12–16)	18	15	12
ALl (4–40 iu.L^{-1})	40	—	—
AST (11–45 iu.L^{-1})	120	—	—
Creatine kinase (16–300 iu.L^{-1})	1202	—	—
LDH (105–195 iu.L^{-1})	475	—	—

non-specific changes in liver, lungs and brain, compatible with the clinical diagnosis of heat-stroke complicated by DIC.

DISCUSSION

Heat stroke is the most severe manifestation of exercise-induced heat illness and is characterized by:

(1) a very high initial core temperature: a figure of >41°C is usually accepted (Kains *et al.*, 1983; Khogali & Al Khawashki, 1981; Malamud *et al.*, 1946; Costrini *et al.*, 1979).

(2) associated neurological disturbances, including headache, drowsiness, confusion, ataxia, delirium and coma. Our case was comatose on arrival as were 10 of the 13 cases reported by Beller & Boyd (1975). Profound rigidity and tonic contractions may occur.

(3) absent sweating: the skin is usually hot and dry but may be cold, as in our case, due to peripheral vasoconstriction (Kains *et al.*, 1983; Khogali & Al Khawashki, 1981).

The homeostatic mechanism of heat transfer from the body is primarily dependent upon sweating and becomes relatively inefficient in conditions of high environmental humidity and temperature. During exercise under these conditions the circulating blood volume falls appreciably (Lawrence & Rubel, 1983) and triggers an over-riding cutaneous vasoconstrictor reflex, possibly barorecepter mediated, leading to reduced heat loss through the skin. This would appear to be the primary pathophysiological mechanism of heat stroke. Similarly, an increase in blood volume is likely to be the primary physiological change in acclimitization to a hot humid environment (Lawrence & Rubel, 1983; Nadel, 1984). Extreme heat (>42°C) denatures enzymes and adversely affects all cell membranes leading to the characteristic biochemical features of heat stroke (Beller & Boyd, 1975). These include: acidosis; hyperkalaemia; myoglobinuria and elevated transaminases. Hypoglycaemia, as in our patient, is also a common feature (Malamud *et al.*, 1946; Beller & Boyd, 1975) and may be exacerbated by pre-exercise starvation.

Disturbances of coagulation are a frequent finding in heat stroke, brought about by a combination of mechanisms including reduced production of clotting factors by the heat damaged liver (Beller & Boyd, 1975), the production of tissue thromboplastins from heat disrupted cells (Convertino *et al.*, 1980) and impaired platelet function (Stefanini & Spicer., 1971). The treatment of disseminated intravascular coagulation in heat stroke remains controversial and the importance of expert haematological advice is emphasized.

The cornerstone of the treatment of heat stroke is its early recognition followed by rapid effective cooling. There is abundant evidence that the survival rate in the condition is a function not only of the degree of pyrexia but also of its duration (Costrini *et al.*, 1979). In the armed services, it is essential that all those responsible for the supervision of physical training should be aware, not only of the mere existence of heat stroke as a disease, but of its potentially fatal outcome.

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